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Hypoxylon Canker of Aspen

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Hypoxylon canker, caused by the fungus *Hypoxylon pruinatum* (Klotsche) Cke., is the most important killing disease of aspen. In the Lake States, where it is particularly damaging, its total impact is estimated to be equivalent to one-third of the net annual growth. Only the decay fungi cause more damage.

Range

The disease is present throughout much of the range of aspen. It occurs in the eastern United States and Canada, west to British Columbia, and south to Arizona. It is reportedly present in Russia, but has not been found in Western Europe.

Hosts

The fungus most commonly infects quaking aspen (*Populus tremuloides* Michx.) (fig. 1); infection levels exceeding 25 percent of the trees in a stand have been reported. Bigtooth aspen (*P. grandidentata* Michx.) is infected occasionally. Infection of balsam poplar (*P. balsamifera* L.) is rare. European aspen (*P. tremula* L.), Chinese aspen (*P. adenopoda* Maxim.), and Bolleana poplar (*P. alba var. pyramidalis*) are additional hosts for the fungus.

Symptoms

Young cankers first appear on aspen bark as slightly sunken yellowish-orange areas with an irregular margin. As infection progresses, the outer bark is raised in blisterlike patches and sloughs off, exposing the blackened crumbling cortex. Old cankers, which may be several feet long, are rough and blackened at the center and yellowish-orange at the newly invaded margins.

Although callus may develop occasionally at the margins of a canker, the fungus usually invades new tissue so rapidly that callus has no time to form. Many trees infected on the lower bole are girdled and killed within 5 years. An infection on the upper bole may cause only part of the crown to die, but the entire tree may then die from suppression. Some trees are so weakened by decay in the cankered zone that wind breaks the stems before girdling is complete. (fig. 2).

The most reliable field symptom is the laminated or mottled black

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Figure 1.—Hypoxylon canker on quaking aspen.

and yellowish-white cortex, which can be exposed by cutting into young cankers or cutting near the margins of older cankers. Removing the bark exposes white mycelial fans in the cambial zone (fig. 3).

Description and Life History of the Fungus

How the hypoxylon canker fungus infects aspen is not definitely known. Most cankers originate in the immediate area of a dead branch stub or break in the bark, but there is no evidence to indicate that the fungus penetrates through the dead tissue. Apparently, airborne spores, after entering through wounds in living bark, germinate to produce mycelium, which invades and kills the surrounding tissue. Insect wounds are reported to be commonly associated with infection. Failures to induce infection arti-

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Figure 2.—Aspen broken by wind at the point where hypoxylon cankers occurred.

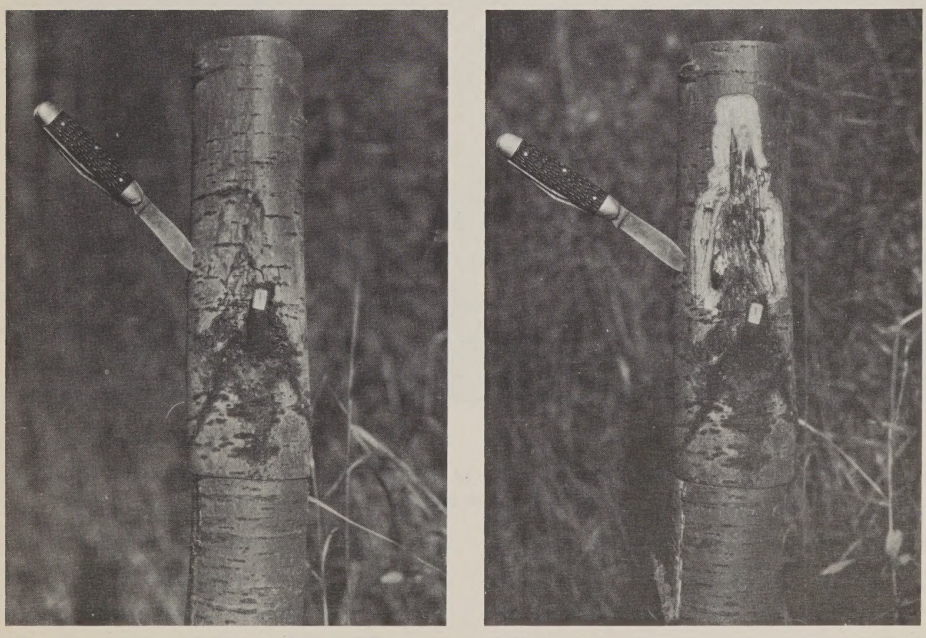

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Figure 3.—*A*, Young Hypoxylon canker above dead branch stub, outer bark beginning to blister and crack. *B*, Bark removed to reveal white mycelial fans at margins and the black mottling of the infected area.

ficially by inoculating wounds with spores indicate that factors *other* than the presence of a wound are also important. Some reports suggest bark moisture levels and chemical or physical characteristics may be related to infectibility of the host.

Five to 14 months after infection, asexual spores are produced. These gray, powdery spores appear annually, from spring until early August, on small bristlelike structures under the blistered outer bark of the younger portions of the canker (fig. 4).

About 3 years after infection, sexual spores are produced in fruiting bodies on the older portions of the canker. The fruiting bodies are small crustlike growths ranging from a few to several millimeters in diameter (fig. 5). When young, they are covered with a grayish bloom, which soon disappears. As the fruiting bodies age, they turn black and appear carbonaceous. When the fruiting bodies get wet, the spores are forcibly discharged. Sporulation can continue for several years after the host tree dies.

Habits

Prevalence of the disease varies from one geographical area to another for reasons not known at present. It also varies within a single area, with high levels of infection occurring in occasional "wave" years. Although disease incidence does not seem to vary with site quality, severity is apparently greater on poor sites. Poor stocking apparently creates conditions favorable to the fungus. Canker causes only about half as many stem losses in well-stocked stands as in poorly stocked ones, and about two to three times as many trees are infected near exposed stand edges as within well-stocked parts of the same stand.

Juvenile trees are most susceptible to infection, but appreciable losses from the disease can occur in older stands. New cankers on mature trees are usually on the upper bole. Dominant, vigorous trees and suppressed, weak trees are both susceptible to infection. There does, however, appear to be a genetic relationship in that some clones have a lower percentage of infected trees.

Control

No direct control measures are known. The general distribution and prevalence of the fungus and the lack of knowledge on the distance spores are carried through the air indicate that sanitation measures would be costly and of questionable value. The few such attempts to eliminate the disease from a stand have been unsuccessful.

In severely infected, poorly-stocked stands, losses attributable to canker may equal or exceed growth. These stands should be harvested at once; if cutting is deferred, the merchantable volume may fall below that considered operable. If partial cuts are made in better stocked aspen stands, all infected trees should be removed

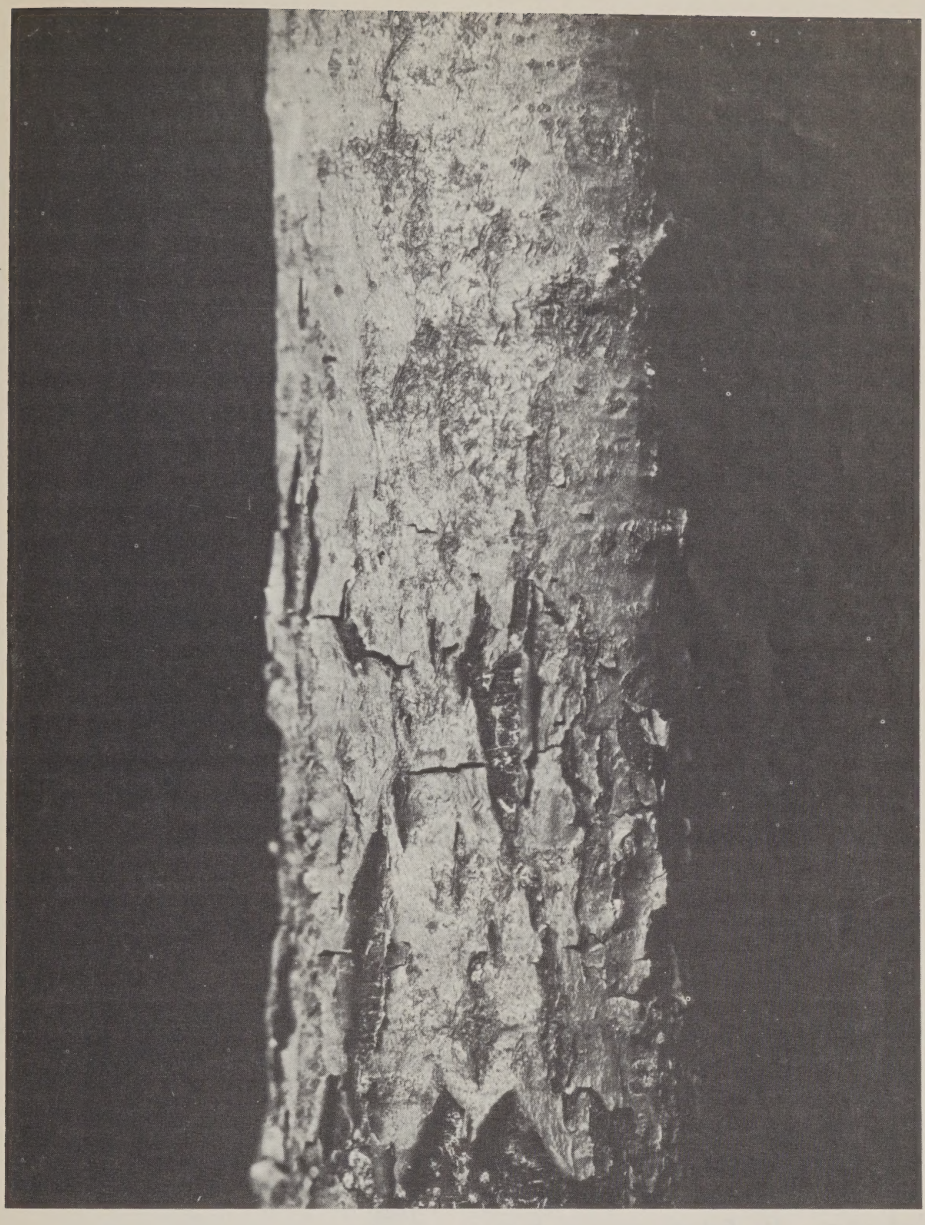

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Figure 4.—Blistered and ruptured outer bark of an aspen stem exposing an inner layer where bristlelike structures bearing asexual spores are produced.

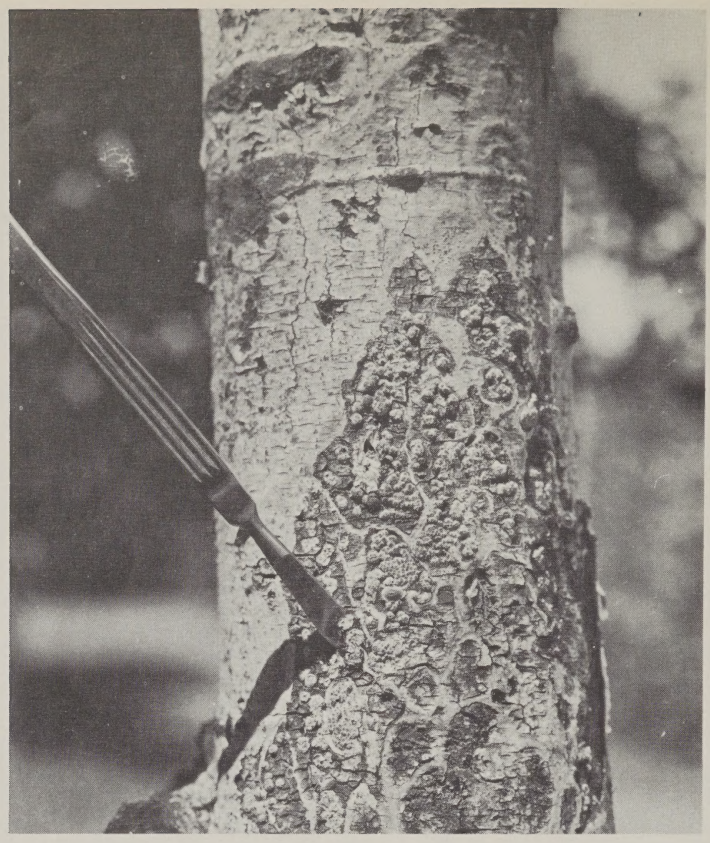

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Figure 5.—Grayish fruiting bodies within which sexual spores are produced. After the second year these structures turn black.

as they would die within 3 to 5 years anyway and their volume lost.

In established stands, there is little opportunity to eliminate exposed stand edges or increase stocking as a means of preventing infection. In future stands, however, losses from canker probably could be reduced if regeneration efforts are directed toward obtaining a well-stocked stand without openings and poorly stocked patches. A uniformly well-stocked stand in a general management objective for all forest types. In aspen stands, reduction of canker losses should be an added incentive for achieving this objective.

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